Restenosis in gold-coated renal artery stents

Brian W. Nolan, MD, Marc L. Schermerhorn, MD, Richard J. Powell, MD, Erin Rowell, MD, Mark F. Fillinger, MD, Eva M. Rzucidlo, MD, Mark C. Wyers, MD, David Whittaker, MD, Robert M. Zwolak, MD, Daniel B. Walsh, MD, and **Jack L. Cronenwett, MD,** *Lebanon, NH*

Background: **Gold coating improves stent visibility under fluoroscopy. This is particularly valuable for precise stent placement during renal artery stenting (RAS). There is conflicting evidence regarding restenosis with gold-coated stents. To evaluate the effect of gold coating on restenosis after renal stenting, we reviewed the results of all patients undergoing RAS in our practice.**

Methods: **A retrospective cohort study of all patients undergoing RAS between June 2000 and September 2003 was performed. During this time, both gold-coated and stainless steel stents were used. Restenosis (>60% diameter) was determined by serial follow-up duplex exams (peak systolic velocity >180cm/s and renal-aortic ratio >3.5). Restenosis rates were determined by using the Kaplan-Meier life-table method. Variables potentially affecting restenosis were evaluated with the log-rank test and Cox proportional hazards modeling.**

Results: **RAS was performed in 97 arteries (78 patients). Gold-coated (NIRoyal) stents were placed in 59 arteries (48 patients). Stainless steel stents (Corinthian, Genesis, and Herculink) were placed in 38 arteries (34 patients). Patient demographics, indication for treatment, technical success, and complications did not differ between gold and stainless steel stent groups. Mean follow-up was 15 months for gold-coated stents and 18 months for stainless steel stents (NS). By life-table method, 1-year and 2-year freedom from restenosis rates were 84% and 78% in arteries treated with stainless steel stents versus 69% and 39% in those treated with gold-coated stents ($P = .012$, log-rank test). By multivariate analysis, only the use of gold-coated stents ($P = .018$; hazard ratio [HR], 3.3; 95% confidence interval [CI], 1.2 to 8.7) and bilateral disease ($P = .046$; HR, 2.3; 95% CI, 1.02 to 5.2) predicted restenosis. Stent diameter, patient demographics, and indication for RAS had no effect on restenosis by univariate analysis. According to American Heart Association criteria, 87% of patients in the stainless steel group had improved blood pressure at 1 year, compared with 77% in the gold-coated stent group (Kaplan-Meier; $P = .042$, log-rank test). There were no significant differences in the effect of RAS on serum creatinine levels between the two groups.**

Conclusion: **Gold-coated renal stents had a substantially higher rate of restenosis than stainless steel stents in our series. These findings have led us to abandon the use of gold-coated stents for RAS. Patients who have received gold-coated stents for the treatment of atherosclerotic renal artery stenosis should be followed closely for evidence of restenosis. (J Vasc Surg 2005;42:40-6.)**

Renal artery percutaneous transluminal angioplasty (RPTA) was introduced by Gruntzig in 1978.[1] Initial reports demonstrated an improvement in blood pressure control in patients with renovascular hypertension and renal function in patients with chronic renal insufficiency.[2-4] The technical success of RPTA was poor, however, and 1-year restenosis was 27% to 100%.[5]

Metallic stents were introduced for the treatment of renal artery stenosis in 1991.[6] Compared with RPTA, renal artery stenting (RAS) has since been shown to improve technical success; however, restenosis rates remain widely variable, from 13% to 43% at 1 year.[7-12] Stent design has continued to evolve during the last 2 decades in an attempt to improve technical success and reduce complication and restenosis rates.

In 1996, gold-coated stents became available for clinical use in the coronary circulation.[13] Gold coating made stents more radio-opaque to enhance visibility under fluoroscopy. These stents subsequently became popular for use in the renal artery, where precise stent placement is required for the treatment of atherosclerotic ostial stenosis. Along with superior visibility, preliminary animal studies also suggested that gold might reduce the incidence of acute stent thrombosis.[14]

Clinical evidence from the coronary circulation, however, has indicated that gold-coated stents have a higher rate of in-stent restenosis secondary to intimal hyperplasia. Three prospective, randomized trials comparing gold-coated and noncoated stents used in coronary arteries demonstrated a higher rate of restenosis in gold-coated stents.[15-17] This problem has not been widely studied for RAS. A single retrospective analysis showed no difference between restenosis in gold-coated and stainless steel stents used to treat renal artery stenosis.[18] The purpose of the this study was to analyze our experience with restenosis in gold-coated versus stainless steel stents used in the treatment of renal artery stenosis.

METHODS

Patients and indications. All patients with atherosclerotic renal artery stenoses who underwent RAS by vascular surgeons at Dartmouth-Hitchcock Medical Center between June 2000 and September 2003 were reviewed. A

From the Division of Vascular Surgery, Dartmouth-Hitchcock Medical Center.

Competition of interest: none.

Presented at the Thirty-first Annual Meeting of the New England Society for Vascular Surgery, Whitefield, NH, Sept 17-19, 2004.

Reprint requests: Brian W. Nolan, MD, Dartmouth-Hitchcock Medical Center, Division of Vascular Surgery, One Medical Center Drive, Lebanon, NH 03756 (e-mail: Brian.Nolan@Hitchcock.org)

0741-5214/$30.00

doi:10.1016/j.jvs.2005.03.038

total of 78 patients (97 arteries) were treated, with follow-up data collected through September 2004. Four patients (5%) were treated with a gold-coated stent on one side and stainless steel stent on the other. Clinical outcomes data (blood pressure and serum creatinine values) from these four patients were omitted from analysis, but stent restenosis data was included.

All patients with bilateral disease who were treated with the same stent type were treated either during the same procedure or within several days to weeks after. Staged treatment was done to minimize contrast exposure. These patients all had an initial diagnosis of bilateral disease (ie, none had a renal artery stenosis detected during follow-up of a contralateral stent). Clinical outcomes and restenosis data in patients with bilateral disease treated with the same type of stent were therefore included in the analysis.

Patients with a clinical indication for treatment of renal artery stenosis were selected in accordance with the American Heart Association (AHA) guidelines.[19,20] Indications included (1) renovascular hypertension either resistant to treatment with at least three medications of different classes or associated with medication intolerance or a solitary kidney, (2) chronic renal insufficiency (serum creatinine '1.4 mg/dL) of unknown etiology, or both.

Arteriography was performed on patients with a clinical indication for therapy in whom a stenosis was identified by duplex scan (peak systolic velocity >200 cm/s or renal-aortic ratio >3.5).[21] Patients with renal insufficiency were pretreated with 600 mg oral N-acetylcysteine twice the day before the procedure and again on the day of the procedure.

Criteria for therapy at angiography was a systolic pressure gradient ≥15 mm Hg or a >75% stenosis. High-grade stenoses (>75% diameter reduction) were stented without measuring a pressure gradient to reduce the risk of embolization. Occluded arteries were revascularized if the ipsilateral kidney was not atrophic (<9 cm) and there was evidence that the occlusion was relatively acute (ie, artery patent by preoperative imaging found to be occluded at angiography). *Bilateral disease* was defined as a right and left renal artery stenosis meeting criteria for therapy or a renal artery stenosis meeting the indications for therapy with a contralateral renal artery occlusion.

Stent selection. During the 3 years studied, three discrete periods occurred during which three types of low profile (0.014" or 0.018" platform) balloon expandable stents were deployed:

- The initial 25 arteries were treated with "over the wire" (OTW) stainless steel stents, either Corinthian or Herculink (Guidant).
- The next 59 arteries were treated with OTW, NIRoyal gold-coated stents (Boston Scientific Corp). The NIRoyal stent became the stent of choice because of its superior visualization that made it technically easier to use and more accurate for the treatment of ostial stenoses.
- The final 13 arteries reported in this series were treated with a Genesis monorail, stainless steel stent (Cordis). The change back to a stainless steel stent was because of its availability in the monorail platform, making it easier to use with the PercuSurge Guardwire (Medtronic) embolic protection device.

Although the type of stent deployed changed with time, all surgeons in the group used the same type of stent during each given time period.

Technique. Percutaneous access was obtained through the common femoral artery unless contraindicated by occlusive or aneurysmal disease, in which case a left brachial approach was used. A micropuncture technique (20-gauge access needle, 0.018-in wire and 5F sheath) was used to minimize access trauma. A 0.035-in guidewire was advanced under fluoroscopic guidance (OEC 9800, 12-in image intensifier or fixed GE system, 16-in image intensifier) into the abdominal aorta. Diagnostic arteriography was performed through a 5F flush catheter. If a renal artery stenosis was confirmed, a 6F left internal mammary artery guiding catheter was positioned at the origin of the renal artery over the 0.035-in wire, taking care not to occlude or touch the orifice of the artery. Intravenous heparin (100 U/kg) was administered. The renal artery was selected and the lesion crossed by using a 0.014-in or a 0.018-in wire.

Embolic protection was employed selectively by using the 0.014-in Guardwire temporary balloon occlusion and aspiration system. Indications for embolic protection were a serum creatinine level of ≥2.0 mg/dL, or a solitary kidney, or both.

Critical lesions (>75%) were predilated with a low-profile balloon (3 × 20 mm). Atherosclerotic lesions were then preferentially treated with balloon-expandable stents. After deployment, a pressure measurement was obtained across the stent using a 4F glide catheter. If a residual gradient >5 mm Hg was present, it was localized then treated with either angioplasty or a second stent. Technical adequacy (stenosis <30%) was also assessed by arteriography.

Postoperatively, all patients were given a 300-mg loading dose of Plavix (Bristol-Myers Squibb Co) and treated for 1 month with a standard 75-mg dose along with 325 mg of aspirin.

Outcomes. Surveillance renal duplex for restenosis was performed 1 month after the procedure and every 3 to 6 months thereafter. Velocity criteria for restenosis were a peak systolic velocity >180 cm/s and renal-aortic ratio >3.5 on follow-up exam.[9] Indications for follow-up arteriogram in patients with velocity elevations by duplex scan included recurrent hypertension or worsening renal function, the presence of a solitary kidney, or clear progression of restenosis determined by subsequent duplex exams. Patients with recurrent stenosis by duplex exam but no other indication for treatment were followed with duplex ultrasound scanning until an indication developed.

Clinical measures of blood pressure and renal function are reported at last follow-up (1-year mean) in accordance

Table I. Demographics and indications for patients undergoing renal stent placement according to stent type—gold-coated or stainless steel

	Gold (n = 44)%	*Steel(n = 30)%*	*P*
Age (years)	69 ± 10	67 ± 15	.74
Female gender	43	50	.72
Diabetes	18	20	.93
Current smoking	85	79	.73
Coronary artery disease	50	47	.64
Hypercholesterolemia	60	67	.66
Peripheral vascular disease	41	40	.88
Abdominal aortic aneurysm	28	30	.82
Bilateral renal artery disease	45	37	.25
Indication for treatment			
Hypertension	50	53	.79
Chronic renal insufficiency	50	47	.64

Table II. Characteristics of renal artery stenoses by duplex and arteriography according to stent type—gold-coated or stainless steel

	Gold (n = 59)	*Steel (n = 38)*	*P*
Duplex			
Peak systolic velocity (cm/s)	316 ± 160	329 ± 135	.71
Renal aortic ratio	4.4 ± 2.4	5.0 ± 2.6	.28
Resistive Index	0.80 ± 0.1	0.81 ± 0.1	.78
Arteriogram			
Ostial location	85%	94%	.14
Occlusions	2	1	NS
Systolic pressure gradient (mm Hg)	52 ± 31	58 ± 40	.63

NS = Not significant.

Table III. Characteristics of renal artery stent procedures by type of stent used—gold-coated or stainless steel

	Gold (n=59)	*Steel (n=38)*	*P*
Stent diameter			.96
3 mm	1.7%	2.7%	
4 mm	10.1%	8.1%	
5 mm	47.5%	51.4%	
6 mm	40.7%	35.1%	
7 mm	0.0%	2.7%	
Stents per artery	1.03	1.08	.37
Fluoroscopy time (minutes)	31 ± 20	36 ± 23	.37
Contrast volume used (mL)	81 ± 53	91 ± 52	.42

with AHA standards.[19,20] *Improved blood pressure* was defined as a diastolic pressure <90 or systolic pressure <140, or both, on the same or a reduced number of medications. Inability to meet these criteria was defined as *failure*. *Improved renal function* was defined as a decrease in serum creatinine levels ≥20% relative to baseline. Renal function was considered *stable* if the creatinine remained ≤20% of baseline and *worse* if it was increased by ≥20%.

Statistical analysis. Dichotomous variables are presented as proportions and compared with the χ^2test. Continuous variables are presented as mean ± standard deviation and compared with the Student's *t* test. Predictors of restenosis were determined by Cox regression with univariate and multivariate analysis. Hypertension response, renal function response, and restenosis rates were analyzed by using Kaplan-Meier life-table methods. Comparisons of these outcomes between groups were made with the log-rank test. $P < .05$ was considered statistically significant.

RESULTS

Ninety-seven arteries were treated for atherosclerotic occlusive disease (3 occlusions, 94 stenoses). Gold-coated stents were used to treat 59 arteries in 48 patients, and 38 arteries in 34 patients received stainless steel stents. Four patients with bilateral disease received both a steel and gold-coated stent. Overall, 50% of patients were treated for renovascular hypertension and 50% for chronic renal insufficiency. Patient demographics and indication for treatment did not differ between gold-coated and stainless steel stent groups (Table I). Duplex and angiographic findings also did not differ statistically between groups (Table II).

Primary technical success was achieved in 92 patients (95%) and did not differ by stent group. Technical failure in the remaining five patients was because of an inability to select the renal artery, usually secondary to acute caudal angulation. These five patients underwent subsequent attempts via the left brachial artery, four of which were successful for a secondary technical success of 98%.

Embolic protection was used in 25 patients: 20 (33%) of 59 gold-coated stents, and 5 (13%) of 38 steel stents. The difference is statistically significant and reflects the fact that the embolic protection device did not become available for our use until early 2002, midway through the series.

Most arteries were treated with 4-, 5-, and 6-mm stents, with no differences in stent diameter between the gold-coated and stainless steel groups. Although slightly more contrast was used on average in the stainless steel group, the difference was not statistically significant (Table III).

There were no perioperative deaths and no operative conversions. Complications occurred in 6 patients (7.3%) and did not differ by group. One minor complication (1.2%) of a groin hematoma did not require operative therapy or blood transfusion but did prolong hospital stay by 1 day for observation. Five major complications (6.1%) occurred:

- Postoperative azotemia (serum creatinine level increase >20%) occurred in three patients (3.9%). Azotemia resolved spontaneously in one patient and after a stent was placed in a contralateral stenosis on post-op day 1 in another. One patient with a baseline

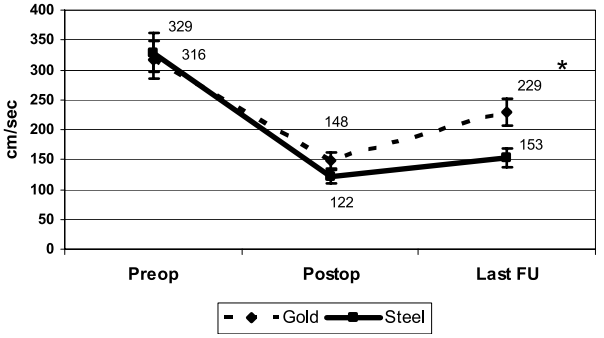

Fig 1. Mean peak systolic velocities measured pre-op, 1-month post-op, and at last follow-up (gold 15 ± 4.5 months, steel 18 ± 7.5 months) in renal arteries treated with gold-coated and stainless steel stents (*$P = .044$, t test).

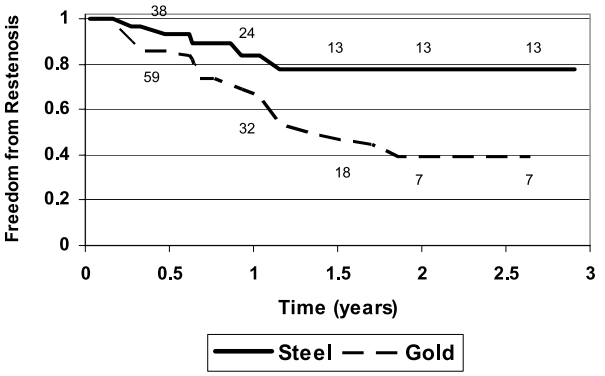

Fig 2. Freedom from restenosis based on surveillance duplex scanning according to stent type—gold-coated or stainless steel (Kaplan-Meier method, standard error <10% throughout, number of patients at risk included with curves; $P = .012$, log rank).

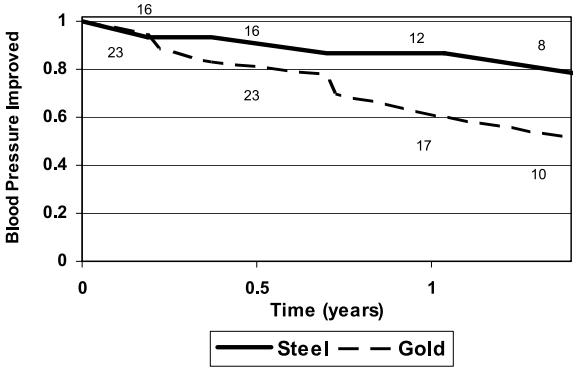

Fig 3. Blood pressure improvement at last follow-up according to stent type—gold-coated or stainless steel (Kaplan-Meier method, standard error <10% to 1-year, number of patients at risk included with curves; $P = .042$, log rank).

serum creatinine value of 2.5 mg/dL developed acute renal failure after a failed attempt at stenting through femoral access. A stent was subsequently placed via a left brachial approach but the patient did not improve and went on to require dialysis.

- In one patient, a perinephric hematoma developed after a parenchymal perforation with a guidewire. This was detected by computed tomography scan after the patient reported flank pain postoperatively. He was transfused with 2 units of packed red blood cells and was discharged on post-op day 5 without further incident.
- One patient had a myocardial infarction and required urgent percutaneous transluminal angioplasty of a coronary artery bypass graft stenosis.

Follow-up duplex data were available on 75 patients (96%) and 92 arteries (95%). One patient was lost to follow-up. Two patients died before follow-up duplex exams: one died 1 month post-op after a motor vehicle accident; the other, who had extensive coronary and peripheral vascular disease, died 4 months after renal artery stenting (1 month after a below-knee amputation) presumably of myocardial infarction.

Follow-up ranged from 1 to 38 months, with an overall mean of 16 months (gold-coated stent group, 15 months; stainless steel group, 18 months, NS). Mean duplex velocities in the steel and gold-coated stent groups at the preoperative, first follow-up at 1 month after operation, and at the last follow-up visit, are depicted in Fig 1. The initial peak systolic velocities at 1 month did not differ significantly between the groups. At the last follow-up, velocities were significantly higher in the gold-coated stent group (229 cm/s vs 153 cm/s; $P = .044$, t test).

Overall, the 1-, 2- and 3-year life-table freedom from restenosis was 75%, 55%, and 55%. In the stainless steel stent group, the 1- and 2-year freedom from restenosis was 84% and 78%. In the gold-coated stent group, the 1- and 2-year freedom from restenosis was 69% and 39% ($P = .012$, log-rank test) (Fig 2).

By multivariate analysis, only bilateral disease ($P = .046$; hazard ratio [HR], 2.3; 95% confidence interval [CI], 1.02 to 5.2) and gold coating ($P = .018$; HR, 3.3; 95% CI, 1.2 to 8.7) were predictors of restenosis. By univariate analysis, indication for treatment (hypertension or chronic renal insufficiency), patient demographics (age, gender, diabetes mellitus, tobacco use, coronary artery disease, peripheral vascular disease, hypercholesterolemia, presence of an abdominal aortic aneurysm), stent diameter (4, 5, or 6 mm), and use of embolic protection, did not correlate with restenosis.

According to AHA criteria, patients in the stainless steel group had better hypertension control than those treated with a gold-coated stent. By life-table analysis, 87% of patients in the stainless steel stent group had improved blood pressure control at 1 year compared with 77% of patients in the gold-coated stent group ($P = .042$, log rank) (Fig 3). There were no significant differences in the effect on serum creatinine levels between the two groups (Fig 4). The overall reintervention rate based on clinical

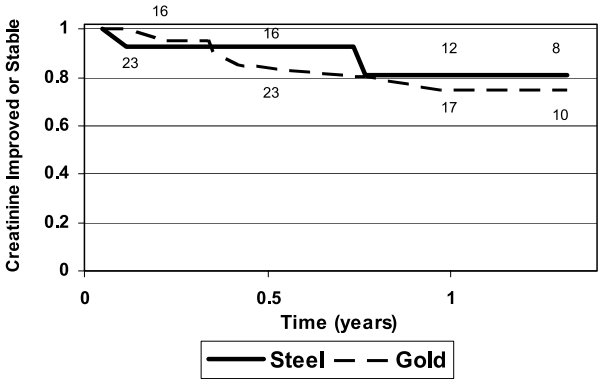

Fig 4. Serum creatinine stable or improved at last follow-up according to stent type—gold-coated or stainless steel (Kaplan-Meier method, standard error <10% to 1-year, number of patients at risk included with curves).

indications was 10% at 1 year and did not differ significantly between groups.

DISCUSSION

The current study compared restenosis in NIRoyal gold-coated stents with several stainless steel stents used to treat atherosclerotic renal artery stenosis. Our results show that a recurrent stenosis was 3.3 times more likely to develop with the gold-coated NIRoyal stents and that patients treated with gold-coated stents had worse hypertension control than patients with stainless steel stents. These findings are consistent with several prospective, randomized studies demonstrating a higher rate of early (6-month) restenosis and intimal hyperplasia in coronary arteries treated with gold-coated versus noncoated stents:

- Park et al[17] reported a nearly twofold higher incidence of angiographic restenosis in 110 coronary arteries randomized to receive gold-coated NIR stents. In this study, there was a trend toward increased major adverse cardiac events in the gold-coated stent group, but the difference was not statistically significant ($P = .113$).[17]
- In 103 patients randomized to receive gold-coated coronary stents, vom Dahl et al[15] demonstrated a 40% greater loss of in-stent luminal diameter by follow-up arteriography at 6 months. However, no differences were noted in clinical outcomes between the two groups in this study.[15]
- In a series of >700 randomized patients, Kastrati et al[16] reported a 1.3-fold higher incidence of angiographic restenosis in gold-coated stents and a significantly less-favorable event-free survival at 1 year ($P = .001$).[16]

In contrast to these results in coronary arteries, a retrospective analysis by Zeller et al[18] reported similar restenosis rates in gold-coated and stainless steel stents used in the treatment of 219 ostial renal artery stenosis.[18] Gold-coated stents were placed in 29% of arteries and stainless steel stents in 71%. The authors found no significant difference in the percentage of patients in the two groups who developed restenosis at 12 months (12.2% in gold coated stents, 11.1% in noncoated stents). However, restenosis was not analyzed using life-table analysis technique, which is different from our study. Also, the criteria used to determine restenosis were not stated. It is possible that some asymptomatic restenosis in the Zeller study were missed if routine follow-up duplex scans or arteriography was not performed. In the Zeller study, logistic regression found only smaller stent diameter predicted restenosis. In our series, no difference was found in restenosis in 4-, 5-, and 6-mm stents using Cox regression.

Overall at our institution, renal duplex scanning is very reliable in detecting renal artery stenosis. Although this was not studied during the same time period of the current series, it has been determined in a previous review using the same criteria (renal-aortic ratio >3.5 or a peak systolic velocity >200 cm/s). These data were submitted for the laboratory's Intersocietal Commission for the Accreditation of Vascular Laboratories certification.

Comparison was performed between noninvasive duplex data and arteriographic evaluation of the same patients from 1995 to 2000. For detection of a hemodynamically significant (>60%) stenosis by arteriogram, renal duplex was 88% sensitive and 92% specific, with a positive predictive value of 93%, a negative predictive value of 85%, and an overall accuracy of 89%. The criteria for restenosis in the current series were also based on duplex velocity measurements.

Although data have suggested elevated duplex velocities in other stented arteries such as carotids, this was not our experience in the renal arteries.[22] Lower velocity criteria were used in defining restenosis because our 1-month postoperative data suggested that velocities are initially low after stenting. The mean velocities at 1 month after stent placement were 148 cm/s in the gold-coated group and 122 cm/s in the steel group (difference not significant) (Fig 1).

Given the initial normalization of velocities, we believed that increasing velocities were a suggestive of restenosis. For example, if a patient were to have an initial post-op velocity of 140 and then return with a velocity of 190, we would consider that evidence of restenosis even though the velocity did not reach the 200-cm/s cutoff that is used in initial screening. The specific reason 180 cm/s was chosen was that it is the only other velocity criteria referenced in the literature for restenosis. This kept us consistent with that report.[9] To date, we have not found any false-positives on repeat arteriography, although we have yet not reintervened on all patients with recurrent velocity elevations.

Patients with gold-coated stents were found to have significantly worse blood pressure control by 1 year. Early loss of blood pressure control is consistent with physiologically significant recurrent stenosis secondary to intimal hyperplasia. Our reintervention rate did not differ between the two groups, however.

In our initial experience, our approach toward asymptomatic restenosis was conservative. Patients who had not experienced obvious clinical deterioration but had restenosis determined by a follow-up duplex scan were typically watched and not taken for repeat angiography until symptoms had recurred. Thus, the finding of worse blood pressure control in the gold-coated stent group suggests to us that we should have intervened sooner in those patients with duplex evidence of restenosis. Our group has subsequently become more aggressive about early reintervention for restenosis, even in stable patients with restenosis determined from duplex exam alone.

One limitation of our series was that the stent strut design was not the same for gold-coated and stainless steel stents. It is thus possible that stent design, rather than gold coating, caused the differences that we observed. However, all the stents in the series, including the various stainless steel stents, differed slightly in strut design, whereas the one clear distinguishing characteristic of the NIRoyal stent is its gold coating.

Biologically, the higher restenosis rate in gold-coated stents has several possible explanations. Gold demonstrates excellent resistance to corrosion in air, but its resistance in the intravascular environment is approximately 100 times less than that of high-grade stainless steel. In vitro studies suggest that corrosion, leading to the diffusion of metal ions into biologic tissues, initiates an inflammatory response.[23] Klein et al[24] has shown that metal ions can upregulate cellular adhesion molecules involved in intimal hyperplasia. Gold also has a higher electronegative surface charge than stainless steel.[25] It is plausible that this difference may contribute to an enhanced cellular inflammatory response within the media that acts as a stimulus for intimal hyperplasia and restenosis.

Finally, it has been suggested the increased hyperplasia in gold-coated NIR stents may be the result of a rougher stent surface resulting from the manufacturing process of applying the gold coating. Edelman et al[14] found that in porcine coronary arteries, the effect on hyperplasia could be abrogated by heating gold-coated stents to alter the surface finish. Although a refined heat treatment processes has improved the surface smoothness of the NIRoyal stent, texture may still have a role in restenosis.

Restenosis remains a significant challenge in the endovascular management of symptomatic renal artery stenosis. Despite this, RAS has largely replaced operative therapy because of lower morbidity and mortality rates. As stent designs evolve to improve technical success and reduce complications and restenosis, it remains important to monitor the results of various stent types, particularly those coated with bioreactive substances.

In our series, gold-coated stents had a significantly higher incidence of restenosis and led to a worsened clinical outcome with respect to blood pressure control. Given these results, further evaluation—ideally a prospective randomized trial comparing gold-coated and stainless steel stents for treatment of symptomatic renal artery stenosis—is warranted. We recommend that patients who have received gold-coated stents for the treatment of atherosclerotic renal artery stenosis be followed closely for evidence of restenosis.

REFERENCES

1. Gruntzig A, Kuhlmann U, Kaufman S. Treatment of renovascular hypertension with percutaneous transluminal dilatation of a renal artery stenosis. Lancet 1978;1:801-2.
2. Katzen B, Chang J, Lukowsky G, Abramson E. Percutanous transluminal angioplasty for the treatment of renovascular hypertension. Radiology 1979;131:53-8.
3. Tegtmeyer C, Kellum C, Ayers C. Percutaneous transluminal angioplasty of the renal artery. Results and long-term follow-up. Radiology 1984;153:77-84.
4. Weibull H, Tornquist C, Bergqvist D, Nyman U, Takolander R, Karlsson S, et al. Reversible renal insufficiency after percutaneous transluminal angioplasty (PTA) of renal artery stenosis. Acta Chir Scand 1984; 150:295-300.
5. Leertouwer TC, Gussenhoven EJ, Bosch JL, van Jaarsveld BC, van Dijk LC, Deinum J, et al. Stent placement for renal arterial stenosis: where do we stand? A meta-analysis. Radiology 2000;216:78-85.
6. Lederman R, Mendelsohn F, Santos R, Phillips H, Stack R, Crowley J. Primary renal artery stenting: characteristics and outcomes after 363 procedures. Am H J 2001;142:314-23.
7. Bakker J, Goffette P, Henry M, Mali WP, Melki JP, Moss JG et al. The Erasme Study: a multicenter study on the safety and technical results of the Palmaz stent used for the treatment of atherosclerotic ostial renal artery stenosis. Cardiovasc Intervent Radiol 1999;22:468-74.
8. Gill K, Fowler R. Atherosclerotic renal arterial stenosis: clinical outcomes of stent placement for hypertension and renal failure. Radiology 2003;226:821-6.
9. Yutan E, Glickerman DJ, Caps MT, Hatsukami T, Harley JD, Kohler TR, et al. Percutaneous transluminal revascularization for renal artery stenosis: Veterans Affairs Puget Sound Health Care System experience. J Vasc Surg 2001;34:685-93.
10. Bush R, Najibi S, MacDonald M, Lin PH, Chaikof EL, Martin LG, et al. Endovascular revascularization of renal artery stenosis: technical and clinical results. J Vasc Surg 2001;33:1041-9.
11. van de Ven PJ, Kaatee R, Beutler JJ, Beek FJ, Woittiez AJ, Buskens E, et al. Arterial stenting and balloon angioplasty in ostial atherosclerotic renovascular disease: a randomised trial. The Lancet 1999;353:282-6.
12. Cognet F, Garcier JM, Dranssart M, Defraissinette B, Cercueil JP, Ravel A, et al. Percutaneous transluminal renal angioplasty in atheroma with renal failure: long-term outcomes in 99 patients. Eur Radiol 2001;11: 2524-30.
13. Harding SA, McKenna CJ, Flapan AD, Boon NA. Long-term clinical safety and efficacy of NIRoyal vs. NIR intracoronary stent. Cathet Cardiovasc Interv 2001;54:141-5.
14. Hehrlein C, Metz J, Ensinger W, Kubler W. Influence of surface texture and change on the biocompatibility of endovascular stents. Coron Artery Dis 1995;6:581-6.
15. vom Dahl J, Haager PK, Grube E, Gross M, Beythien C, Kromer EP , et al. Effects of gold coating of coronary stents on neointimal proliferation following stent implantation. Am J Cardiol 2002;89:801-5.
16. Kastrati A, Schomig A, Dirschinger J, Mehilli J, von Welser N, Pache J, et al. Increased risk of restenosis after placement of gold-coated stents: results of a randomized trial comparing gold-coated with uncoated steel stents in patients with coronary artery disease. Circulation 2000;101: 2478-83.
17. Park SJ, Lee CW, Hong MK, Kim JJ, Park SW, Tahk SJ, et al. Comparison of gold-coated NIR stents with uncoated NIR stents in patients with coronary artery disease. Am J Cardiol 2002;89:872-5.
18. Zeller T, Muller C, Frank U, Burgelin K, Sinn L, Horn B, et al. Gold coating and restenosis after primary stenting of ostial renal artery stenosis. Cathet Cardiovasc Interv 2003;60:1-60.
19. Rundback JH, Sacks D, Kent KC, Cooper C, Jones D, Murphy T, et al. Guidelines for the reporting of renal artery revascularization in clinical trials. J Vasc Interv Radiol 2002;13:959-74.

20. Rundback JH, Sacks D, Kent KC, Cooper C, Jones D, Murphy T, et al. Guidelines for the reporting of renal artery revascularization in clinical trials. Circulation 2002;106:1572-1585.
21. Olin JW, Piedmonte MR, Young JR, DeAnna S, Grubb M, Childs MB. The utility of duplex ultrasound scanning of the renal arteries for diagnosing significant renal artery stenosis. Ann Intern Med 1995;122: 833-8.
22. Lal BK, Hobson I, Robert W., Goldstein J, Chakhtoura EY, Duran WN. Carotid artery stenting: is there a need to revise ultrasound velocity criteria? J Vasc Surg 2004;39:58-66.
23. Steinemann SG. Metal implants and surface reactions. Injury 1996;27: sc16-22.
24. Klein CL, Nieder P, Wagner M, Kohler H, Bittinger F, Kirkpatrick CJ, et al. The role of metal corrosion in inflammatory processes: induction of adhesion molecules by heavy metal ions. J Materials Sci 1994;5:798-807.
25. Sprague EA, Palmaz JC, Simon C, Watson A. Electrostatic forces on the surface of metals as measured by atomic force microscopy. J Long Term Eff Med Implants 2000;10:111-25.

Submitted Nov 30, 2004; accepted Mar 26, 2005.